```
     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:29:24 ON 27 MAY 2005
L1          5886 S CHROMATIN (S) REMODEL?
L2        373850 S FUSION OR CHIMER?
L3         16399 S "CHROMATIN STRUCTURE"
L4         18906 S DNA (3W) "BINDING DOMAIN"
L5          4729 S "ZINC FINGER" (S) DOMAIN
L6           722 S CHROMATIN (5W) MODIFICATION
L7        381389 S WOLFF?/AU OR SMITH?/AU
L8             0 S SANGAMO?/AU
L9           176 S SANGAMO?
L10        49251 S PROTEIN (3W) COMPLEX
L11          169 S L10 AND L1
L12            3 S L11 AND L4
L13            3 DUP REM L12 (0 DUPLICATES REMOVED)
L14          352 S L2 AND L1
L15           39 S L14 AND L4
L16           13 S L15 NOT PY>=2001
L17            7 DUP REM L16 (6 DUPLICATES REMOVED)
L18            5 S L7 AND L2 AND L1
L19            3 DUP REM L18 (2 DUPLICATES REMOVED)
L20        32480 S URNOV?/AU OR PABO?/AU OR HOLMES?/AU
L21           36 S L20 AND L3
L22           17 S L21 NOT PY>=2001
L23            9 DUP REM L22 (8 DUPLICATES REMOVED)
L24          371 S TARGETED (S) CHROMATIN
L25           12 S L24 AND L20
L26           35 S L24 AND (L7 OR L9)
L27            7 S L25 NOT PY>=2001
L28           12 S L26 NOT PY>=2001
L29            3 DUP REM L27 (4 DUPLICATES REMOVED)
L30            7 DUP REM L28 (5 DUPLICATES REMOVED)
L31          255 S RECOMBINATION AND (L6 OR L1)
L32            3 S L31 AND L2
L33            1 DUP REM L32 (2 DUPLICATES REMOVED)
L34         1018 S L5 (P) L4
L35            0 S L34 AND L24
L36          189 S L34 AND L2
L37           12 S L34 AND L1
L38            6 DUP REM L37 (6 DUPLICATES REMOVED)
L39            2 S L38 NOT PY>=2001
L40            2 S L36 AND L1
L41            1 DUP REM L40 (1 DUPLICATE REMOVED)

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L41 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002161958 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11893498
TITLE: Biotechnologies and therapeutics: chromatin as a target.
AUTHOR: Reik Andreas; Gregory Philip D; Urnov Fyodor D
CORPORATE SOURCE: Sangamo Biosciences, Pt Richmond Tech Center, 501 Canal Blvd, Suite A100, Richmond, California 94804, USA.
SOURCE: Current opinion in genetics & development, (2002 Apr) 12 (2) 233-42. Ref: 108
Journal code: 9111375. ISSN: 0959-437X.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020315
Last Updated on STN: 20020606
Entered Medline: 20020605

AB As alterations in gene expression underlie a considerable proportion of human diseases, correcting such aberrant transcription in vivo is expected to provide therapeutic benefit to the patient. Attempts to control endogenous mammalian genes, however, face a significant obstacle in the form of chromatin. Aberrant gene repression can be alleviated by using small-molecule inhibitors that exert nucleus-wide effects on chromatin-based repressors. Genome-wide **chromatin remodeling** also occurs during cloning via nuclear transfer, and causes the deregulation of epigenetically controlled genes. Regulation of genes in vivo can be accomplished via the use of designed transcription factors - these result from a **fusion** of a designed **DNA -binding domain** based on the **zinc finger** protein motif to a functional **domain** of choice.

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L13 ANSWER 1 OF 3 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 2004284999 EMBASE
TITLE: The DNA-binding properties of the ARID-containing subunits of yeast and mammalian SWI/SNF complexes.
AUTHOR: Wilsker D.; Patsialou A.; Zumbrun S.D.; Kim S.; Chen Y.; Dallas P.B.; Moran E.
CORPORATE SOURCE: E. Moran, Fels Institute for Cancer Research, Temple University School of Medicine, Phialdelphia, PA, United States. betty@temple.edu
SOURCE: Nucleic Acids Research, (2004) Vol. 32, No. 4, pp. 1345-1353.
Refs: 37
ISSN: 0305-1048 CODEN: NARHAD
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
022 Human Genetics
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040722
Last Updated on STN: 20040722
AB SWI/SNF complexes are ATP-dependent **chromatin remodeling** complexes that are highly conserved from yeast to human. From yeast to human the complexes contain a subunit with an ARID (A-T-rich interaction domain) **DNA-binding domain**. In yeast this subunit is SWI1 and in human there are two closely related alternative subunits, p270 and ARID1B. We describe here a comparison of the DNA-binding properties of the yeast and human SWI/SNF ARID-containing subunits. We have determined that SWI1 is an unusual member of the ARID family in both its ARID sequence and in the fact that its DNA-binding affinity is weaker than that of other ARID family members, including its human counterparts, p270 and ARID1B. Sequence analysis and substitution mutagenesis reveals that the weak DNA-binding affinity of the SWI1 ARID is an intrinsic feature of its sequence, arising from specific variations in the major groove interaction site. In addition, this work confirms the finding that p270 binds DNA without regard to sequence specificity, excluding the possibility that the intrinsic role of the ARID is to recruit SWI/SNF complexes to specific promoter sequences. These results emphasize that care must be taken when comparing yeast and higher eukaryotic SWI/SNF complexes in terms of DNA-binding mechanisms.

L13 ANSWER 2 OF 3 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 97247142 EMBASE
DOCUMENT NUMBER: 1997247142
TITLE: Steroid receptor induction of gene transcription: A two-step model.
AUTHOR: Jenster G.; Spencer T.E.; Burcin M.M.; Tsai S.Y.; Tsai M.-J.; O'Malley B.W.
CORPORATE SOURCE: B.W. O'Malley, Department of Cell Biology, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030, United States
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 15, pp. 7879-7884.
Refs: 44
ISSN: 0027-8424 CODEN: PNASA6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 970904
Last Updated on STN: 970904
AB Coactivators, such as steroid receptor coactivator 1 (SRC-1A) and CREB (cAMP response element binding protein)-binding protein (CBP), are

required for efficient steroid receptor transactivation. Using an in vitro transcription assay, we found that progesterone receptor (PR)-driven transcription is inhibited by a dominant negative PR ligand-binding domain- interacting region of SRC-1A, indicating that SRC-1A is required for actual transcriptional processes. In addition, these coactivators also possess intrinsic historic acetyltransferase (HAT) activity and bind to each other and another HAT, p300/CBP-associated factor. Here we show that the human PR also interacts with p300/CBP-associated factor in vitro. Recruitment of multiple HATs to target promoters suggests an important role for **chromatin remodeling** in transcriptional activation of genes by steroid receptors. In transient transfection assays, we found that addition of a histone deacetylase inhibitor, trichostatin A, strongly potentiated PR-driven transcription. In contrast, directing histone deacetylase-1 (HD1) to a promoter using the GAL4 ***DNA*** **binding domain** inhibited transcription. Furthermore, PR transactivation was repressed by recruiting HD1 into the PR- DNA complex by fusing HD1 to a PR ligand-binding domain-interacting portion of SRC-1. Collectively, these results suggest that targeted historic acetylation by recruited HAT cofactors and histone deacetylation are important factors affecting PR transactivation. Recruitment of coactivators and HATs by the liganded PR in vivo may result in (i) **remodeling** of transcriptionally repressed **chromatin** to facilitate assembly and (ii) enhanced stabilization of the preinitiation complex by the activation functions of coactivators and the liganded PR itself.

L13 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:271282 BIOSIS
DOCUMENT NUMBER: PREV199800271282
TITLE: Role of nucleosome **remodeling** factor NURF in transcriptional activation of **chromatin**.
AUTHOR(S): Mizuguchi, Gaku; Tsukiyama, Toshio; Wisniewski, Jan; Wu, Carl [Reprint author]
CORPORATE SOURCE: Lab. Mol. Cell Biol., Natl. Cancer Inst., Natl. Inst. Health, Build. 37, Room 5E-26, Bethesda, MD 20892-4255, USA
SOURCE: Molecular Cell, (Dec., 1997) Vol. 1, No. 1, pp. 141-150. print.
ISSN: 1097-2765.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998

AB The Drosophila nucleosome remodeling factor (NURF) is a **protein complex** of four subunits that assists transcription factor-mediated perturbation of nucleosomes in an ATP-dependent manner. We have investigated the role of NURF in activating transcription from a preassembled chromatin template and have found that NURF is able to facilitate transcription mediated by a GAL4 derivative carrying both a DNA binding and an activator domain. Interestingly, once nucleosome remodeling by the DNA binding factor is accomplished, a high level of NURF activity is not continuously required for recruitment of the general transcriptional machinery and transcription for at least 100 nucleotides. Our results provide direct evidence that NURF is able to assist gene activation in a chromatin context, and identify a stage of NURF dependence early in the process leading to transcriptional initiation.

=>

L17 ANSWER 1 OF 7 MEDLINE on STN
ACCESSION NUMBER: 2000270200 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10809742
TITLE: Peptides selected to bind the Gal80 repressor are potent transcriptional activation domains in yeast.
AUTHOR: Han Y; Kodadek T
CORPORATE SOURCE: Departments of Internal Medicine and Biochemistry, Center for Biomedical Inventions, Ryburn Center for Molecular Cardiology, University of Texas Southwestern Medical Center, Dallas, Texas 75235-8573, USA.
CONTRACT NUMBER: P50 HL55988 (NHLBI)
SOURCE: Journal of biological chemistry, (2000 May 19) 275 (20) 14979-84.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000629
Last Updated on STN: 20000629
Entered Medline: 20000621

AB The activation domain of the yeast Gal4 protein binds specifically to the Gal80 repressor and is also thought to associate with one or more coactivators in the RNA polymerase II holoenzyme and **chromatin remodeling** machines. This is a specific example of a common situation in biochemistry where a single protein domain can interact with multiple partners. Are these different interactions related chemically? To probe this point, phage display was employed to isolate peptides from a library based solely on their ability to bind Gal80 protein in vitro. Peptide-Gal80 protein association is shown to be highly specific and of moderate affinity. The Gal80 protein-binding peptides compete with the native activation domain for the repressor, suggesting that they bind to the same site. It was then asked if these peptides could function as activation domains in yeast when tethered to a **DNA binding domain**. Indeed, this is the case. Furthermore, one of the Gal80-binding peptides binds directly to a domain of the Gal11 protein, a known coactivator. The fact that Gal80-binding peptides are functional activation domains argues that repressor binding and activation/coactivator binding are intimately related properties. This peptide library-based approach should be generally useful for probing the chemical relationship of different binding interactions or functions of a given native domain.

L17 ANSWER 2 OF 7 MEDLINE on STN
ACCESSION NUMBER: 1999262964 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10330133
TITLE: The activity of mammalian brm/SNF2alpha is dependent on a high-mobility-group protein I/Y-like **DNA binding domain.**
AUTHOR: Bourachot B; Yaniv M; Muchardt C
CORPORATE SOURCE: Unite des Virus Oncogenes, URA1644 du CNRS, Departement des Biotechnologies, Institut Pasteur, Paris, France.
SOURCE: Molecular and cellular biology, (1999 Jun) 19 (6) 3931-9.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990628
Last Updated on STN: 19990628
Entered Medline: 19990617

AB The mammalian SWI-SNF complex is a **chromatin-remodelling** machinery involved in the modulation of gene expression. Its activity relies on two closely related ATPases known as brm/SNF2alpha and BRG-1/SNF2beta. These two proteins can cooperate with nuclear receptors for transcriptional activation. In addition, they are involved in the

control of cell proliferation, most probably by facilitating p105(Rb) repression of E2F transcriptional activity. In the present study, we have examined the ability of various brm/SNF2alpha deletion mutants to reverse the transformed phenotype of ras-transformed fibroblasts. Deletions within the p105(Rb) LXCXE binding motif or the conserved bromodomain had only a moderate effect. On the other hand, a 49-amino-acid segment, rich in lysines and arginines and located immediately downstream of the p105(Rb) interaction domain, appeared to be essential in this assay. This region was also required for cooperation of brm/SNF2alpha with the glucocorticoid receptor in transfection experiments, but only in the context of a reporter construct integrated in the cellular genome. The region has homology to the AT hooks present in high-mobility-group protein I/Y DNA binding domains and is required for the tethering of brm/SNF2alpha to chromatin.

L17 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:536359 BIOSIS
DOCUMENT NUMBER: PREV199900536359
TITLE: A conserved motif N-terminal to the DNA-binding domains of myogenic bHLH transcription factors mediates cooperative DNA binding with Pbx-Meis1/Prep1.
AUTHOR(S): Knoepfler, Paul S.; Bergstrom, Don A.; Uetsuki, Taichi; Dac-Korytko, Ia; Sun, Y. Henry; Wright, Woodring E.; Tapscott, Stephen J.; Kamps, Mark P. [Reprint author]
CORPORATE SOURCE: Department of Pathology, School of Medicine, University of California-San Diego, 9500 Gilman Drive, La Jolla, CA, 92093, USA
SOURCE: Nucleic Acids Research, (Sept. 15, 1999) Vol. 27, No. 18, pp. 3752-3761. print.
CODEN: NARHAD. ISSN: 0305-1048.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1999
Last Updated on STN: 10 Dec 1999
AB The t(1;19) chromosomal translocation of pediatric pre-B cell leukemia produces **chimeric** oncoprotein E2a-Pbx1, which contains the N-terminal transactivation domain of the basic helix-loop-helix (bHLH) transcription factor, E2a, joined to the majority of the homeodomain protein, Pbx1. There are three Pbx family members, which bind DNA as heterodimers with both broadly expressed Meis/Prep1 homeodomain proteins and specifically expressed Hox homeodomain proteins. These Pbx heterodimers can augment the function of transcriptional activators bound to adjacent elements. In heterodimers, a conserved tryptophan motif in Hox proteins binds a pocket on the surface of the Pbx homeodomain, while Meis/Prep1 proteins bind an N-terminal Pbx domain, raising the possibility that the tryptophan-interaction pocket of the Pbx component of a Pbx-Meis/Prep1 complex is still available to bind tryptophan motifs of other transcription factors bound to flanking elements. Here, we report that Pbx-Meis1/Prep1 binds DNA cooperatively with heterodimers of E2a and MyoD, myogenin, Mrf-4 or Myf-5. As with Hox proteins, a highly conserved tryptophan motif N-terminal to the DNA-binding domains of each myogenic bHLH family protein is required for cooperative DNA binding with Pbx-Meis1/Prep1. In vivo, MyoD requires this tryptophan motif to evoke **chromatin remodeling** in the Myogenin promoter and to activate Myogenin transcription. Pbx-Meis/Prep1 complexes, therefore, have the potential to cooperate with the myogenic bHLH proteins in regulating gene transcription.

L17 ANSWER 4 OF 7 MEDLINE on STN
ACCESSION NUMBER: 1999096944 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9878427
TITLE: The glutamine-rich domain of the Drosophila GAGA factor is necessary for amyloid fibre formation in vitro, but not for **chromatin remodelling**.
AUTHOR: Agianian B; Leonard K; Bonte E; Van der Zandt H; Becker P B; Tucker P A
CORPORATE SOURCE: Structural Biology Programme, European Molecular Biology Laboratory, Meyerhofstrasse 1 D-69117, Heidelgberg, Germany.

SOURCE: Journal of molecular biology, (1999 Jan 15) 285 (2) 527-44.
Journal code: 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990324
Last Updated on STN: 19990324
Entered Medline: 19990311

AB The Drosophila GAGA factor binds specifically to the sequence GAGAG, and synergises with nucleosome **remodelling** factor to **remodel** **chromatin** in vitro. It consists of an N-terminal domain (POZ/BTB) which mediates protein-protein interactions, a central region which contains the **DNA-binding domain**, and a C-terminal glutamine-rich region. It is shown that the glutamine-rich region is responsible for the formation of fibres in vitro which, on the basis of their tinctorial properties and CD spectra, may be classified as amyloid fibres. A large structural change, probably resulting in beta-sheet structure, is observed upon fibre formation. Mutants containing the central region, either alone or together with the glutamine-rich region, are largely lacking in secondary structure but they bind specifically to the cognate DNA and are able to **remodel** **chromatin** in vitro. Consequently, neither the N-terminal domain nor the C-terminal glutamine-rich regions of the GAGA factor are necessary for **chromatin remodelling** in vitro.

L17 ANSWER 5 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1999069418 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9852087
TITLE: Mutations in the AF-2/hormone-binding domain of the **chimeric** activator GAL4.estrogen receptor.VP16 inhibit hormone-dependent transcriptional activation and **chromatin remodeling** in yeast.
AUTHOR: Stafford G A; Morse R H
CORPORATE SOURCE: Molecular Genetics Program, Wadsworth Center, New York State Department of Health, and State University of New York School of Public Health, Albany, New York 12201-2002, USA.
CONTRACT NUMBER: GM51993 (NIGMS)
SOURCE: Journal of biological chemistry, (1998 Dec 18) 273 (51) 34240-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990209
Last Updated on STN: 19990209
Entered Medline: 19990126

AB GAL4.estrogen receptor.VP16 (GAL4.ER.VP16), which contains the GAL4 **DNA-binding domain**, the human ER hormone binding (AF-2) domain, and the VP16 activation domain, functions as a hormone-dependent transcriptional activator in yeast (Louvion, J.-F., Havaux-Copf, B., and Picard, D. (1993) Gene (Amst.) 131, 129-134). Previously, we showed that this activator can **remodel** **chromatin** in yeast in a hormone-dependent manner. In this work, we show that a weakened VP16 activation domain in GAL4.ER.VP16 still allows hormone-dependent **chromatin remodeling**, but mutations in the AF-2 domain that abolish activity in the native ER also eliminate the ability of GAL4.ER.VP16 to activate transcription and to **remodel chromatin**. These findings suggest that an important role of the AF-2 domain in the native ER is to mask the activation potential of the AF-1 activation domain in the unliganded state; upon ligand activation, a conformational change releases AF-2-mediated repression and transcriptional activation ensues. We also show that the AF-2 domain, although inactive at simple promoters on its

own in yeast, can enhance transcription by the MCM1 activator in hormone-dependent manner, consistent with its having a role in activation as well as repression in the native ER.

L17 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1999110097 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9894806
TITLE: Recruitment of the RNA polymerase II holoenzyme and its implications in gene regulation.
AUTHOR: Barberis A; Gaudreau L
CORPORATE SOURCE: Institute of Molecular Biology, University of Zurich, Switzerland.
SOURCE: Biological chemistry, (1998 Dec) 379 (12) 1397-405. Ref: 82
Journal code: 9700112. ISSN: 1431-6730.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990317

AB In yeast cells, interaction between a DNA-bound protein and a single component of the RNA polymerase II (poIII) holoenzyme is sufficient to recruit the latter to a promoter and thereby activate gene transcription. Here we review results which have suggested such a simple mechanism for how genes can be turned on. The series of experiments which eventually led to this model was originally instigated by studying gene expression in a yeast strain which carries a point mutation in Gal11, a component of the poIII holoenzyme. In cells containing this mutant protein termed Gal11P, a derivative of the transcriptional activator Gal4 devoid of any classical activating region is turned into a strong activator. This activating function acquired by an otherwise silent DNA-binding protein is solely due to a novel and fortuitous interaction between Gal11P and a fragment of the Gal4 dimerization region generated by the P mutation. The simplest explanation for these results is that tethering Gal11 to DNA recruits the poIII holoenzyme and, consequently, activates gene transcription. Transcription factors that are believed not to be integral part of the poIII holoenzyme but are nevertheless required for this instance of gene activation, e.g. the TATA-binding TFIID complex, may bind DNA cooperatively with the holoenzyme when recruited to a promoter, thus forming a complete poIII preinitiation complex. One prediction of this model is that recruitment of the entire poIII transcription complex and consequent gene activation can be achieved by tethering different components to DNA. Indeed, **fusion** of a **DNA-binding domain** to a variety of poIII holoenzyme components and TFIID subunits leads to activation of genes bearing the recognition site for the DNA-binding protein. These results imply that accessory factors, which are required to remove or modify nucleosomes do not need to be directly contacted by activators, but can rather be engaged in the activation process when the poIII complex is recruited to DNA. In fact, recruitment of the poIII holoenzyme suffices to remodel nucleosomes at the PHO5 promoter and presumably at many other promoters. Other events in the process of gene expression following recruitment of the transcription complex, e.g. initiation, promoter clearance, elongation and termination, could unravel as a consequence of the recruitment step and the formation of an active preinitiation complex on DNA. This view does not exclude the possibility that classical activators also act directly on **chromatin remodeling** and post-recruitment steps to regulate gene expression.

L17 ANSWER 7 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 97269067 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9111067
TITLE: **Chromatin remodeling** by transcriptional activation domains in a yeast episome.

AUTHOR: Stafford G A; Morse R H
CORPORATE SOURCE: Molecular Genetics Program, Wadsworth Center, New York State Department of Health and State University of New York School of Public Health, Albany, New York 12201-2002, USA.
CONTRACT NUMBER: GM51993 (NIGMS)
SOURCE: Journal of biological chemistry, (1997 Apr 25) 272 (17) 11526-34.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970602
Last Updated on STN: 19970602
Entered Medline: 19970521
AB We examine the generality of transcription factor-mediated **chromatin remodeling** by monitoring changes in **chromatin** structure in a yeast (Saccharomyces cerevisiae) episome outside of the context of a natural promoter. The episome has a well defined chromatin structure and a binding site for the transcription factor GAL4 but lacks a nearby functional TATA element or transcription start site, so that changes in chromatin structure are unlikely to be caused by transcription. To separate changes caused by binding and by activation domains, we use both GAL4 and a **chimeric**, hormone-dependent activator consisting of the GAL4 **DNA-binding domain**, an estrogen receptor (ER) hormone-binding domain, and a VP16 activation domain (Louvion, J.-F., Havaux-Copf, B. and Picard, D. (1993) Gene (Amst.) 131, 129-134). Both GAL4 and GAL4.ER.VP16 show very little perturbation of chromatin structure in their nonactivating configurations. Substantial additional perturbation occurs upon activation. This additional perturbation is marked by changes in micrococcal nuclease cleavage patterns, restriction endonuclease accessibility, and DNA topology and is not seen with the nonactivating derivative GAL4.ER. Remodeling by GAL4.ER.VP16 is detectable within 15 min following hormone addition and is complete within 45 min, suggesting that replication is not required. We conclude that activation domains can exert a major influence on **chromatin remodeling** by increasing binding affinity and/or by recruitment of other **chromatin remodeling** activities and that this **remodeling** can occur outside the context of a bona fide promoter.

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L30 ANSWER 1 OF 7 MEDLINE on STN
ACCESSION NUMBER: 2000133586 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10668407
TITLE: Thyroid hormone receptor, v-ErbA, and chromatin.
AUTHOR: **Wolffe A P**; Collingwood T N; Li Q; Yee J; Urnov F; Shi Y B
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, Bethesda, Maryland 20892-5431, USA.
SOURCE: Vitamins and hormones, (2000) 58 449-92. Ref: 197
Journal code: 0413601. ISSN: 0083-6729.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000314
Last Updated on STN: 20000314
Entered Medline: 20000301

AB The thyroid hormone receptor and the highly related viral oncoprotein v-erbA are found exclusively in the nucleus as stable constituents of chromatin. Unlike most transcriptional regulators, the thyroid hormone receptor binds with comparable affinity to naked and nucleosomal DNA. In vitro reconstitution experiments and in vivo genomic footprinting have delineated the chromatin structural features that facilitate association with the receptor. Chromatin bound thyroid hormone receptor and v-erbA generate Dnase I hypersensitive sites independent of ligand. The unliganded thyroid hormone receptor and v-erbA associate with a corepressor complex containing NCoR, SIN3, and histone deacetylase. The enzymatic activity of the deacetylase and a chromatin environment are essential for the dominant repression of transcription by both the unliganded thyroid hormone receptor and v-erbA. In the presence of ligand, the thyroid hormone receptor undergoes a conformational change that weakens interactions with the corepressor complex while facilitating the recruitment of transcriptional coactivators such as p300 and PCAF possessing histone acetyltransferase activity. The ligand-bound thyroid hormone receptor directs chromatin disruption events in addition to histone acetylation. Thus, the thyroid hormone receptor and v-erbA make very effective use of their stable association with chromatin and their capacity to alter the chromatin environment as a major component of the transcription regulation process. This system provides an exceptionally useful paradigm for investigating the structural and functional consequences of **targeted chromatin** modification.

L30 ANSWER 2 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001114141 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10961924
TITLE: Co-repressor complexes and remodelling chromatin for repression.
AUTHOR: **Wolffe A P**; Urnov F D; Guschin D
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Heath and Human Development, NIH, Building 18T, Room 106, Bethesda, MD 20892-5431, USA.. awlme@helix.nih.gov
SOURCE: Biochemical Society transactions, (2000) 28 (4) 379-86.
Ref: 50
Journal code: 7506897. ISSN: 0300-5127.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010215

AB Recent progress identifies **targeted chromatin**

remodelling by co-repressor complexes as being an integral component of transcriptional silencing. Here we discuss how chromatin structure and the basal transcriptional machinery are manipulated by the co-repressor complex containing the Mi-2 nucleosomal ATPase, the histone-binding protein RbAp48 and histone deacetylase and by the co-repressor complex containing SIN3, RbAp48 and histone deacetylase. Remarkably, both of these complexes also contain methyl-CpG-binding proteins. This observation provides a molecular mechanism to integrate DNA methylation fully into gene control in vertebrates.

L30 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:359857 BIOSIS
DOCUMENT NUMBER: PREV200000359857
TITLE: **Targeted** cross-linking and DNA cleavage within model **chromatin** complexes.
AUTHOR(S): Lee, Kyu-Min [Reprint author]; Chafin, David R.; Hayes, Jeffrey J.
CORPORATE SOURCE: Department of Biochemistry and Biophysics, University of Rochester Medical Center, Rochester, NY, USA
SOURCE: Wassarman, Paul M.; **Wolffe, Alan P**. Methods Enzymol., (1999) pp. 231-251. Methods in Enzymology; Chromatin. print.
Publisher: Academic Press Inc., 525 B Street, Suite 1900, San Diego, CA, 92101-4495, USA; Academic Press Ltd., 24-28 Oval Road, London, NW1 7DX, UK. Series: Methods in Enzymology.
CODEN: MENZAU. ISSN: 0076-6879. ISBN: 0-12-1822-5-2 (cloth).
DOCUMENT TYPE: Book
Book; (Book Chapter)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Aug 2000
Last Updated on STN: 8 Jan 2002

L30 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2000068893 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10601972
TITLE: Nuclear receptors: coactivators, corepressors and chromatin remodeling in the control of transcription.
AUTHOR: Collingwood T N; Urnov F D; **Wolffe A P**
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, National Institutes of Health, Building 18T, Room 106, Bethesda, Maryland 20892-5431, USA.
SOURCE: Journal of molecular endocrinology, (1999 Dec) 23 (3) 255-75. Ref: 174
Journal code: 8902617. ISSN: 0952-5041.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000309
Last Updated on STN: 20000309
Entered Medline: 20000224

AB A contemporary view of hormone action at the transcriptional level requires knowledge of the transcription factors including the hormone receptor that may bind to promoters or enhancers, together with the chromosomal context within which these regulatory proteins function. Nuclear receptors provide the best examples of transcriptional control through the **targeted** recruitment of large protein complexes that modify chromosomal components and reversibly stabilize or destabilize **chromatin**. Ligand-dependent recruitment of transcriptional coactivators destabilizes chromatin by mechanisms including histone acetylation and contacts with the basal transcriptional machinery. In contrast, the recruitment of corepressors in the absence of ligand or in the presence of hormone antagonists serves to stabilize chromatin by the targeting of histone deacetylases. Both activation and repression require

the action of other chromatin remodeling engines of the switch 2/sucrose non-fermentable 2 (SWI2/SNF2) class. Here we summarize this information and integrate hormone action into a chromatin context.

L30 ANSWER 5 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1998327925 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9663395
TITLE: A multiple subunit Mi-2 histone deacetylase from Xenopus laevis cofractionates with an associated Snf2 superfamily ATPase.
AUTHOR: Wade P A; Jones P L; Vermaak D; **Wolffe A P**
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland 20892, USA.
SOURCE: Current biology : CB, (1998 Jul 2) 8 (14) 843-6.
Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF059185
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981008
Last Updated on STN: 20000303
Entered Medline: 19981001

AB Chromatin structure plays a crucial regulatory role in the control of gene expression. In eukaryotic nuclei, enzymatic complexes can alter this structure by both **targeted** covalent modification and ATP-dependent **chromatin** remodeling. Modification of histone amino termini by acetyltransferases and deacetylases correlates with transcriptional activation and repression [1-3], cell growth [4], and tumorigenesis [5]. Chromatin-remodeling enzymes of the Snf2 superfamily use ATP hydrolysis to restructure nucleosomes and chromatin, events which correlate with activation of transcription [6,7]. We purified a multi-subunit complex from Xenopus laevis eggs which contains six putative subunits including the known deacetylase subunits Rpd3 and RbAp48/p46 [8] as well as substoichiometric quantities of the deacetylase-associated protein Sin3 [9-13]. In addition, we identified one of the other components of the complex to be Mi-2, a Snf2 superfamily member previously identified as an autoantigen in the human connective tissue disease dermatomyositis [14,15]. We found that nucleosome-stimulated ATPase activity precisely copurified with both histone deacetylase activity and the deacetylase enzyme complex. This association of a histone deacetylase with a Snf2 superfamily ATPase suggests a functional link between these two disparate classes of chromatin regulators.

L30 ANSWER 6 OF 7 MEDLINE on STN
ACCESSION NUMBER: 97169333 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9081669
TITLE: Histone acetyltransferases in control.
AUTHOR: Wade P A; **Wolffe A P**
CORPORATE SOURCE: Laboratory of Molecular Embryology, NICHHD, Bethesda, Maryland, 20892-5431, USA.. awlme@helix.nih.gov
SOURCE: Current biology : CB, (1997 Feb 1) 7 (2) R82-4. Ref: 13
Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970414
Last Updated on STN: 19970414
Entered Medline: 19970328

AB Several transcriptional regulators have been found to act as enzymes that acetylate histones. The **targeted** post-translational modification of histones within regulatory nucleoprotein complexes provides an attractive mechanism for controlling transcription within a

chromatin environment.

L30 ANSWER 7 OF 7 MEDLINE on STN
ACCESSION NUMBER: 96059357 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7583085
TITLE: Centromeric chromatin. Histone deviants.
AUTHOR: **Wolffe A P**
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, NIH, Bethesda, Maryland 20892-2710, USA.
SOURCE: Current biology : CB, (1995 May 1) 5 (5) 452-4. Ref: 14
Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19960124
Entered Medline: 19951127

AB Highly variant histones are **targeted** to specialized **chromatin** domains, such as the centromere where they have an essential role in the segregation of sister chromatids at mitosis.

=>

L29 ANSWER 1 OF 3 MEDLINE on STN
ACCESSION NUMBER: 2000133586 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10668407
TITLE: Thyroid hormone receptor, v-ErbA, and chromatin.
AUTHOR: Wolffe A P; Collingwood T N; Li Q; Yee J; **Urnov F** ; Shi Y B
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, Bethesda, Maryland 20892-5431, USA.
SOURCE: Vitamins and hormones, (2000) 58 449-92. Ref: 197
Journal code: 0413601. ISSN: 0083-6729.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000314
Last Updated on STN: 20000314
Entered Medline: 20000301

AB The thyroid hormone receptor and the highly related viral oncoprotein v-erbA are found exclusively in the nucleus as stable constituents of chromatin. Unlike most transcriptional regulators, the thyroid hormone receptor binds with comparable affinity to naked and nucleosomal DNA. In vitro reconstitution experiments and in vivo genomic footprinting have delineated the chromatin structural features that facilitate association with the receptor. Chromatin bound thyroid hormone receptor and v-erbA generate Dnase I hypersensitive sites independent of ligand. The unliganded thyroid hormone receptor and v-erbA associate with a corepressor complex containing NCoR, SIN3, and histone deacetylase. The enzymatic activity of the deacetylase and a chromatin environment are essential for the dominant repression of transcription by both the unliganded thyroid hormone receptor and v-erbA. In the presence of ligand, the thyroid hormone receptor undergoes a conformational change that weakens interactions with the corepressor complex while facilitating the recruitment of transcriptional coactivators such as p300 and PCAF possessing histone acetyltransferase activity. The ligand-bound thyroid hormone receptor directs chromatin disruption events in addition to histone acetylation. Thus, the thyroid hormone receptor and v-erbA make very effective use of their stable association with chromatin and their capacity to alter the chromatin environment as a major component of the transcription regulation process. This system provides an exceptionally useful paradigm for investigating the structural and functional consequences of **targeted chromatin** modification.

L29 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001114141 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10961924
TITLE: Co-repressor complexes and remodelling chromatin for repression.
AUTHOR: Wolffe A P; **Urnov F D**; Guschin D
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Heath and Human Development, NIH, Building 18T, Room 106, Bethesda, MD 20892-5431, USA.. awlme@helix.nih.gov
SOURCE: Biochemical Society transactions, (2000) 28 (4) 379-86. Ref: 50
Journal code: 7506897. ISSN: 0300-5127.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010215

AB Recent progress identifies **targeted chromatin**

remodelling by co-repressor complexes as being an integral component of transcriptional silencing. Here we discuss how chromatin structure and the basal transcriptional machinery are manipulated by the co-repressor complex containing the Mi-2 nucleosomal ATPase, the histone-binding protein RbAp48 and histone deacetylase and by the co-repressor complex containing SIN3, RbAp48 and histone deacetylase. Remarkably, both of these complexes also contain methyl-CpG-binding proteins. This observation provides a molecular mechanism to integrate DNA methylation fully into gene control in vertebrates.

L29 ANSWER 3 OF 3 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2000068893 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10601972
TITLE: Nuclear receptors: coactivators, corepressors and chromatin remodeling in the control of transcription.
AUTHOR: Collingwood T N; **Urnov F D**; Wolffe A P
CORPORATE SOURCE: Laboratory of Molecular Embryology, National Institute of Child Health and Human Development, National Institutes of Health, Building 18T, Room 106, Bethesda, Maryland 20892-5431, USA.
SOURCE: Journal of molecular endocrinology, (1999 Dec) 23 (3) 255-75. Ref: 174
Journal code: 8902617. ISSN: 0952-5041.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000309
Last Updated on STN: 20000309
Entered Medline: 20000224

AB A contemporary view of hormone action at the transcriptional level requires knowledge of the transcription factors including the hormone receptor that may bind to promoters or enhancers, together with the chromosomal context within which these regulatory proteins function. Nuclear receptors provide the best examples of transcriptional control through the **targeted** recruitment of large protein complexes that modify chromosomal components and reversibly stabilize or destabilize **chromatin**. Ligand-dependent recruitment of transcriptional coactivators destabilizes chromatin by mechanisms including histone acetylation and contacts with the basal transcriptional machinery. In contrast, the recruitment of corepressors in the absence of ligand or in the presence of hormone antagonists serves to stabilize chromatin by the targeting of histone deacetylases. Both activation and repression require the action of other chromatin remodeling engines of the switch 2/sucrose non-fermentable 2 (SWI2/SNF2) class. Here we summarize this information and integrate hormone action into a chromatin context.

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Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	1979	transcription WITH (modulator or modifier or alter or regulat)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L2	6164	chromatin	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L3	15319	nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L4	254	(nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L5	108	(((nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin) and "zinc finger") and "dna binding"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L6	0	(chromatin WITH remodel) SAME DNMT	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L7	56	(transcription WITH (modulator or modifier or alter or regulat)) SAME chromatin	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L8	5	"6534261".pn. or "6607882".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L9	335	chromatin WITH remodeling	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L10	189	(chromatin WITH remodeling) and ("fusion protein" or "fusion construct")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L11	176	((chromatin WITH remodeling) and ("fusion protein" or "fusion construct")) and (subunit or component or multi-protein or multiprotein)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27

L12	93	(((chromatin WITH remodeling) and ("fusion protein" or "fusion construct")) and (subunit or component or multi-protein or multiprotein)) and "zinc finger"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L13	122	((nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin) and "zinc finger"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L14	46	((((nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin) and "zinc finger") and "dna binding") and "chromatin structure"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L15	3	"6607882".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L16	12	chromatin SAME DNMT	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L17	35	chromatin WITH remodel	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L18	2	(chromatin WITH remodel) SAME (methylase or demethylase or acetylase or deacetylase or helicase)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L19	47921	"fusion protein" or "chimeric protein" or chimera or "fusion construct"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L20	2361	("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L21	931	(("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin) and "DNA binding domain"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L22	403	chromatin WITH remodel$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27

L23	149	(((("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin) and "DNA binding domain") and (chromatin WITH remodel$)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L24	1220	"chromatin structure"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27
L25	78	((((("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin) and "DNA binding domain") and (chromatin WITH remodel$)) and "chromatin structure"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 11:27

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